THE VETERINARY RECORD

FOUNDED BY WILLIAM HUNTING, F.R.C.V.S., 1888

No. 7. FEBRUARY 12, 1949 Vol. 61.

The Relation of Distemper Infection to the Aetiology of Canine Encephalopathies*

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Introduction

The problem of encephalitis in dogs has been inseparably linked with distemper and its apparent complexity has revolved around the question, "What are all the manifestations of canine distemper and how can they be recognised with certainty?" The recent work of MacIntyre, Trevan and Montgomerie (1948) is an outstanding milestone in progress although there is still a long road to travel before we reach the end.

This paper is concerned with the aetiology of canine encephalitis particularly regarding the evidence on the potential role of distemper virus in the causation of demyelination and encephalitis in dogs. It is written chronologically, to some extent, which will indicate how beliefs have arisen based upon the insecure grounds of clinico-pathological observations alone without resource to good experimentation, and how the lack of irrefutable diagnostic criteria of spontaneous distemper infections in dogs (without resource to ferret inoculation) has hampered progress.

1. The Ancient and Clinical Era

Distemper in dogs was well-known to laymen over 100 years ago; in sporting books published about that time "distemper" may be described and will certainly mention ". . . the heavy eyes . . . the dry husky cough . . . the nasty moisture from eyes and nostrils . . . the agitated movements of the limbs so that the dog can barely support himself. . . . obstinate purging . . . wasting . . . shivering . . . the endless varieties of the complaint . . ." and last, but not least . . . "*the convulsive fits.*" These descriptions are all an elaboration of this theme by veterinarians of that era. Those by Youatt (1845) or Blaine (1817)† are classical models of shrewd clinical observation and in effect are hardly improved upon in modern text-books of veterinary medicine.

† "The distemper commences its attack in various ways . . . it is a disease that in its rise, appearances, progress, duration and termination exhibits more varieties than any other complaint . . . the first symptom noticed is violent looseness and scowering, in others an occasional fit . . . gradual wasting . . . slight cough . . . nose and eyes become moister . . . watery moisture soon changes to pus . . . eyes and nose glued up . . . frequent sneezing, but according as one part or another is the principal seat of complaint so do the symptoms vary . . . appearances of fever are usually considerable . . . dull, disinclination for food, occasional sickness . . . varieties are immense . . . in some an universal eruption on the body under the belly, down inside the thighs . . . eruption is pustular . . . frequently attended by convulsions it is also now and then preceded by them . . . two kinds: simple paralytic affection frequently leaves a spasmodic twitching in one or more limbs for life; . . . the other is a perfect fit in which every part of the body becomes convulsed . . . attended by mental alienation . . . when distemper has made some considerable progress and fits come on followed in succession by each other the animal seldom recovers . . . fits may appear trifling at first." (Blaine.)

The stage was thus set for the subsequent traditional belief lasting until now that distemper was a fever with protean manifestations which in its train might bring exanthematous, gastro-enteric, pulmonary *and nervous forms.* On clinical grounds alone before the establishment of aetiology there was no necessity to dispute this. If there was only one febrile disease of dogs with such a varied clinical picture in different animals then diagnosis was easy and it was not considered peculiar that some dogs should show severe nervous dysfunction and others none at all. (One might draw here the analogy in modern medicine of that vague entity the "flu" with its running nose, red streaming eyes, cough, fever, headache, etc, and post-influenzal encephalitis). Pathological contributions on distemper were of little worth, mainly subscribed to what was clinical fact and were largely observations on pathological anatomy made by clinicians. There was in fact no diagnostic pathology.

2. The Aetiological Era

The highly infectious and contagious nature of distemper was recognised over a 100 years ago and a form of "Jennerian" vaccination had some vogue to create immunity. The original identification of the virus nature of the infection was made by Carré (1905) but final clarification came from the classical contributions of Dunkin and Laidlaw (1926). This work was done under rigorously controlled conditions; as a start dogs were bred under quarantine for experimental use. Some of their observations have a cogent importance to the problem of canine encephalitis and some attention thus must be given to what was observed by them. Distemper, as seen by Dunkin and Laidlaw, in experimental dogs, was an acute fever beginning after a three to six day incubation period. The animals showed a diphasic temperature curve, coryza, severe gastro-intestinal disturbance with vomiting, loss of appetite, offensive diarrhoea and other symptoms due to inflammation of the respiratory tract, while exanthematous rash, transient corneal opacity and keratitis often occurred; many animals recovered. Nothing characteristic was found at autopsy, not even frank bronchopneumonia when it was most expected from clinical findings. This syndrome was seen whether infection was by contact, by intranasal, intracutaneous, or subcutaneous routes and was quite independent of the size of infecting inoculum. In many dogs, "chorea," "fits," "epileptiform" convulsions occurred, but there was no pathological substratum to these symptoms in the form of a fulminating encephalitis. In such "nervous cases" these authors observed only a varied degree of *nerve cell damage* scattered widely in the brain. They made no claim to have transmitted an encephalitis in the true sense nor to have recovered any neurotropic virus, least of all distemper, from such cases. Under natural conditions, where spread is by contact, we know that the clinical picture is more often complicated by secondary infection and the commonest one might appear to be that of pneumonia due *B. bronchisepticus* and *streptococci*, etc., or gastro-enteritis. They described the high susceptibility of the ferret to distemper virus and the almost diagnostic course of a coryzal, febrile illness starting after about ten days incubation which often ended fatally. A small proportion of their experimental ferrets showed nervous symptoms (tremors, twitching, screaming fits) many of which showed improvement, but although the words "acute encephalitis" are used, it must have been meant in a clinical sense because the lesions were said to be "neuronal damage" in widely scattered areas of the brain but *without inflammatory* reaction. (In one ferret which had recovered from distemper (and which was immune to subsequent doses of the virus) an acute encephalitis was found but the authors frankly acknowledge that perhaps this was due to another infectious agent.) Later Laidlaw (1930) stated that there was no characteristic pathology in distemper and that it would be impossible to tell from an autopsy whether a dog had died from distemper or not. "It was the custom to allocate all lesions in the nervous system of dogs to distemper infection, but that this could not represent the facts," further that brain tissue from severe canine encephalitic cases did not infect ferrets. All highly important statements from the workers who originally clarified the aetiology of distemper under conditions of experimentation which were rarely observed by subsequent workers.

Many pathologists with an experience of routine dog autopsy

* This paper formed the basis of a talk given at a Symposium on Multiple Sclerosis, American Association for Nervous and Mental Disorders, New York, December 10th, 1948.

work might differ from these opinions on non-specific pathology, although Laidlaw no doubt was basing his views on experimental virus infection. In young dogs, which have suffered from an acute, febrile coryzal, illness, particularly if the temperature was diphasic and which on autopsy show a frank broncho-pneumonia, there would appear to be little doubt of anatomical diagnosis; if sections of nasal mucosa, turbinate bones, bronchial and bladder epithelium in addition showed intracytoplasmic inclusion bodies a diagnosis of distemper infection might be acceptable to all but the most sceptical. It would seem that there is an urgent need for a complete and thorough re-investigation of the pathology* of distemper as a whole and not only from the microscopic aspect. Wharton and Wharton (1934) considered that distemper was essentially an acute anaemia and hypoglycaemia; their paper is noteworthy for the attention paid to clinical-pathological data. The nature, occurrence and distribution of inclusion bodies also requires attention; the work by Watson and Plummer (1942) indicated to some extent their variability and considered that inclusion bodies were much more likely to be found in an animal in a moribund state than in one at the height of illness and that they were an aid to microscopic diagnosis but were not pathognomonic. I have also often failed to find cytoplasmic inclusion bodies in so-called typical clinical distemper and have found them when least of all expecting to find them on the basis of other clinico-pathogical data.

3. The Pathological Era without Experimentation

The general belief, starting 100 years ago at least, therefore grew up that an encephalitic form of canine distemper existed although no association with the "virus" had been proved. [A peculiar fact given to me hearsay, to which little attention has been paid, emphasised by Bloom (private communication) is that dogs fully recovered from distemper rarely develop encephalitis!] The terms "distemper encephalitis," "post-distemper encephalitis," "distemper chorea," have all been loosely applied to canine cases showing *nervous symptoms* simply because at one time or another, the animals had (many had not) suffered from some previous febrile illness which was assumed to be distemper. In view of the lack of knowledge concerning the pathology of canine febrile illnesses this is understandable. Although it seemed that some true canine cases of uncomplicated virus distemper showed "nervous symptoms" it is doubtful if any of these had ever shown the same pathology as that seen in a substantiated encephalitis, no matter what type. Yet most veterinary medicine text-books (*e.g.*, Hutrya and Marek, 1946) state something to the effect that during the septicaemic stage the virus settles in the nervous system where it may cause *inflammatory and degenerative processes;* the general descriptions in such books would leave one with much perplexity on the nature of distemper covering a host of manifestations attached to some febrile disorder.

A form of disseminated encephalomyelitis was recognised in the dog by the pathologist long before the isolation of the virus by Dunkin and Laidlaw (1926). One of the earliest papers was that of Cerletti (1912). A critical analysis of the pathological findings in such papers was made by King and Meehan (1948); it is not the intention to deal with pathology except in so far as it concerns the connection of the distemper virus with neuropathological findings. The principal features described by those older writers such as Cerletti (1912) were meningoencephalitis with a varying degree of severity of focal and perivascular infiltration of plasma cells and lymphocytes, haemorrhage, gliosis, and proliferation of vascular adventitial and endothelial cells. All of which in the light of present day knowledge is concomitant with a virus encephalitis.

It is recognised that a new position was created by the observation of Perdrau and Pugh (1930) when the word "demyelination" was used for the first time in veterinary pathology. They observed demyelination, of a type said to be seen in subacute disseminated sclerosis in man, in four out of 14 canine cases, and admitted that, if a more detailed examination of the nervous system had been made in the others, it might have been found more often. All these dogs had suffered from a febrile illness, at variable previous periods, little different from the symptoms described by Dunkin and Laidlaw (1926) for distemper. They came to the conclusion that in this canine encephalitis the virus of distemper played a similar role to that which the acute infections of varying aetiology played in the causation of certain demyelinating diseases of man. (Laidlaw was unable to recover distemper, or any other, virus by inoculation into ferrets of brain material obtained from some similar cases of Pugh). Demyelination in canine encephalitis was also described by Posrednik (1931), Marinesco *et al.* (1933), Peters and Yamagiwa (1935), and by King (1939) who gave a very complete description of the first case of demyelinating encephalopathy in a dog in the U.S.A. In his case King found widespread lesions in optic tracts and chiasma, cerebellum, hippocampal fissure and cord. The paper is of importance in other directions in discussing the process of demyelination which he said must be distinguished from destruction of nerve tissue as a whole. Goldberg and Volgenau (1925) described a variety of nervous lesions in cases of clinical distemper from which it is difficult to separate pathological type of disease. Apart from demyelination, all the features of canine encephalitis described by previous workers were also given with great neuropathological detail by Gallego (1928). Cordy (1942) describes different types of encephalitic lesions in dogs and divides them into clinical groups; in one group frank inflammatory lesions were present in cases diagnosed clinically as distemper; this paper is noteworthy for the attention given to so-called "old dog encephalitis" which was almost strictly perivascular lymphocytic inflammation and which was said to be non-transmissible to ferrets. The paper by Mettler and Goss (1946) is of great importance for the detailed description of a case of *canine chorea* (probably used in the true clinical sense) in which there was striocerebellar degeneration (hitherto not observed) with absence of perivascular inflammation or demyelination; canine chorea to them was a symptom-complex which might be caused by other factors than the distemper virus unless the latter produced two different types of pathology. The work of Hurst *et al.* (1943) is dealt with later.

The most that could have been said therefore by pathological analysis alone would be that "encephalitis"* in dogs occupied exactly the same peculiar position to distemper as the so-called "post-infective encephalitides" in man which apparently follow various diseases such as small-pox, measles, chicken-pox, whooping cough, influenza, etc. The occurrence of a demyelinating encephalopathy following small-pox and rabies vaccination is in the same category; the latter occurs also in dogs (see von Móczy (1937)) and has still to be explained on an aetiological basis, *i.e.*, whether due to original virus, to the lighting up of another latent neurotropic virus or to an "allergic" effect of heterologous brain emulsion. The work of Kabat *et al.* (1947), is of extreme importance in this direction as acute severe demyelination encephalopathy can be produced in monkeys (and guinea-pigs, see Jarvis and Koprowski (1948)) by a single subcutaneous injection of heterologous, or homologous, brain emulsions with the aid of adjuvants. The "anti-rabic vaccination" encephalitis in dogs can be a very severe disease and is like the condition produced experimentally in monkeys.

4. Personal Investigations

By 1939 nearly 50† dogs had been examined and a complete pathological examination made; all cases, either dead or in a terminal stage of illness, had been sent in by veterinary practitioners and had been diagnosed as suffering from "fits," "chorea," "hysteria," "epilepsy," "post-distemper encephalitis." The *post-mortem* examinations were carried out in all cases by the usual routine procedure to exclude any possible extra-cerebral causes of "nervous derangement"; no single organ in the body was exempt from attention. The brain and parts of the cord were fixed in neutral-formol-saline prior to further examination except in those cases where portions of the brain were preserved fresh for ferret inoculation. Sections were made at many levels from the olfactory lobes to the brain stem and stained by routine and neuropathological methods—some entirely after paraffin embedding, others after celloidin. Many other tissues were taken to provide material for a search for inclusion bodies. For control material there were available sections relating to all organs of hundreds of dogs suffering from a variety of diseases. In a number of cases‡ blood, spleen, liver or parts of the brain were taken, ground up in sterile saline and inoculated into ferrets for distemper virus identification;

* There are no original papers of any merit dealing with the pathology of distemper, and it would seem that most pathological data has been transferred from text-book to text-book.

* This does not include a variety of lesions which have no doubt been included by many as part of "an encephalitis," and which may well have been due to factors other than any virus infection. (cf. The case of Mettler and Goss, and the attention now being given to influence of nutritional factors in nervous syndromes of dogs, Smith (1944)).

† Many more cases were examined in subsequent years but are not included because of the extremely confusing pictures. One outbreak of "distemper," I am sure, included both distemper proper and some other infection, in different dogs in the same kennel.

‡ Some of the material at that time was also sent to the Wellcome Research Laboratories, for ferret inoculation.

this was done with the invaluable help and advice of my chief, T. Dalling (then Professor of Animal Pathology, Cambridge, now Chief Veterinary Officer, Great Britain) who had had a lengthy experience of canine distemper related to the production of distemper vaccine while working at the Wellcome Research Laboratories, Beckenham. By 1939 it was obvious that for any further profitable investigation experimental work was necessary and that this required dogs bred in isolation and more important still, adequate facilities for working with infections in dogs and ferrets; this was impossible and the work was then terminated. Sufficient material had, however, been gathered on which to base a pathological study of canine encephalitis. At that time, Dr. H. J. Scherer was working in the Institut Bunge, Antwerp, and had published a paper (Scherer and Collet, 1939) dealing with the pathology of three cases of canine encephalitis; it was agreed that some collaborative effort might be made in dealing with the comparative neuropathology of canine disorders. All my sections, and the remainder of fixed soft tissues, were therefore sent to him for preliminary study and were lost in the aftermath of war. Suitable acknowledgment in this direction is made in the monograph by Scherer (1944) written before his death. A very brief description of the findings was given by Innes (1940); a more detailed summary is given below based on the protocols written at that time; unfortunately they cannot be supplemented by illustrations, or by a re-examination of the hundreds of sections made, which would have been invaluable in the light of the work of MacIntyre, Trevan and Montgomerie (1948). The various cases had, at that time, been grouped in the following categories:

(*a*) *Distemper and the absence of pathological states in the brain.*—These included dogs which had shown either a febrile illness (distemper?) with "fits," or "fits" without any other apparent illness. Some may have been true distemper, while others may have been examples of true-hysteria now known to be caused by a toxic factor in flour subjected to the "agene" process (see Parry, 1948). No lesions were found in the nervous system. In one case with a history of distemper ferrets inoculated with spleen/blood emulsion died from a distemper-virus infection at the recognised period. There was no visceral pathology present in any of these dogs apart from the occasional case of acute bronchopneumonia.

(*b*) *Distemper and glial clusters in the nervous system.*—In three cases with a history of a recent acute febrile illness (distemper), showing mild fits, glial clusters (as are seen in many toxaemic conditions of man), were found scattered in all parts of the brain; there was no intra-cerebral inflammatory reaction and no visceral pathology was present.

(*c*) *Distemper and Meningitis.*—In two cases with clinical distemper (a diphasic temperature curve, great irritability, tremors, fits, one with corneal opacity) there was a mild lymphocytic meningeal infiltration, mainly basal, without involvement of nervous tissue.

(*d*) *Disseminated demyelinating encephalomyelitis.*—By far the largest group of dogs came within this category; the lesions were comparable to those described by many authors (*e.g.*, Cerletti, Perdrau & Pugh, Peters & Yamigawa, King, Hurst and Scherer & Collet—their Case 2). There was an extremely wide divergence in the severity and distribution of lesions in the different cases; all showed perivascular, and focal lymphocytic and plasma cell infiltrations often sharply delimiting foci of demyelination; many different regions of the nervous system were affected in various cases—cerebrum, basal ganglia, cerebellum, peduncles, pons, optic tracts and chiasma and cord; reticulin proliferation, the presence of "gemästete glia" and gliosis all mentioned by other authors were seen. The age incidence seemed to be over puppyhood, but dogs of all ages were included in the series. Fever was not a constantly recorded clinical symptom, while the variability of the length of the illness starting only sometimes with a distemper-like attack was a very outstanding feature; the periods ranged from two weeks up to three to four months. Fits, intermittency of illness, catarrhal symptoms, various pareses, blindness, vomiting and ataxia, were all mentioned in the clinical reports sent. In some there was a history of an epidemic trend, more than one dog in a kennel being affected; in a few it was mentioned that the administration of anti-distemper serum had no beneficial effect. From one dog at least distemper virus was identified by inoculation of spleen/blood into ferrets, but attempts to identify any virus by inoculation of brain material into ferrets were negative. (In the monograph by Scherer (1944) this type of disease is classified as "*akute multiple Sklerose des Hundes*" and he states that this is the commonest canine encephalopathy, no doubt based upon examination of my own material. This name may, to some, have too close an annotation to disseminated sclerosis in man.)

(*e*) *Cortical proliferative capillaritis.*•—Three cases had a clinical history of a recent acute febrile illness, two had shown fits and one blindness. In each of these there was present an intense vascular proliferation of the small vessels of cortical and subcortical zones of the cerebrum together with marked macroglial reaction. Two of these animals had a terminal acute bronchopneumonia and purulent conjunctivitis. This lesion is described by Scherer and Collet (1939)—their Case 1—as a "pure case"; the three in this series were all present along with an encephalitis described in (*d*). From one of these dogs inoculation of spleen/blood emulsion into ferrets produced an acute distemper-like illness *one month later* (then assumed to be distemper, but now evident from recent work that this might have been some other virus); inoculation of brain material from another case was negative up to the same period.

(*f*) *Diffuse lymphocytic perivascular encephalitis with sclerosis.*—Four cases in this group corresponded with Case 3 described by Scherer and Collet (1939) although each of them had some individual peculiarities: the four cases were all adult (three very old) dogs; in two there was a history of a distemper-like illness at least *two years previously*, with a subsequent periodic, remittent illness, but one always becoming progressively worse, with fits and paralysis. In one the pathological process was universal throughout the brain, in another only the white matter was involved, in another the lesions predominated in the grey matter, but not in cerebellum or cord, while in the fourth perivascular lymphocytic infiltration was almost absent. There was diffuse sclerosis in all and in addition a slight degree of internal hydrocephalus. Injections of spleen/blood and brain from one case into ferrets were negative, while in another inoculation of spleen/blood, but not brain, caused ferrets to go down with a distemper-like illness.

It was concluded, then, in 1939 that a number of pathological entities occurred in the brain of dogs, suffering from "nervous distemper" but in which the association with the distemper virus was problematical; the aetiology was "more likely to be linked with a virus or viruses, not yet identified" (Innes, 1940). It was by reflection along these lines that we (Innes and Dalling, 1939) were attracted to the work of Green *et al.*, who had studied fox encephalitis in the U.S.A. (1929, onwards). It is only necessary to mention relevant observations of Green: (1) A severe form of encephalitis occurred in foxes caused by a filterable virus; (2) this virus was distinct from the distemper variety, but was transmissible to dogs but not to ferrets; (3) true canine distemper occurred in the fox; (4) in fox encephalitis infection transmitted to dogs by cisternal routes, specific inclusions were found in ependymal and endothelial cells of the nervous system (as opposed to the neuronal and neuroglial inclusions of distemper); (5) highly specific intra-nuclear inclusions were also found in hepatic cells. Some preliminary experimental work was therefore done with Dalling with six dogs. Our general conclusions based on this and on my own pathological observations recorded above can be summarised here:

(1) Fox encephalitis virus and distemper virus infections (using Wellcome distemper virus) were separate entities; there was no cross immunity in dogs inoculated with either virus subsequent to recovery from the other infection.

(2) The clinical picture of fever and catarrhal symptoms was alike in the experimental dogs, but a diphasic temperature curve was seen only in distemper.

(3) Fox encephalitis virus injected cisternally into dogs might produce meningitis and ependymitis or an acute encephalitis.

(4) The ependymal, and intranuclear hepatic cell inclusions were seen in experimental fox-encephalitis virus infection in dogs as described by Green.

(5) A type of fox encephalitis had been identified by us in foxes in England; in the two cases seen, the pathological picture showed a symmetry and severity of lesions (localised to temporal lobes), never seen in canine encephalitis. There were, however, no records of natural fox encephalitis infection in dogs in England (but see later reference to work of Rubarth). In the light of the work of MacIntyre *et al.*, it would be most important for such fox cases to be investigated thoroughly.

(6) No intranuclear inclusions in hepatic cells were ever seen in any of my cases of natural canine encephalitis or any form of "nervous distemper"; hence the conclusion then (admittedly based on rather slender evidence) was that the fox encephalitis virus had no connection with any form of "canine encephalitis" as we had seen it.

• This lesion has been described before by other workers as part of a general encephalitic process in dogs, and reference is made by some to its occurrence in lead, CO and thiophen poisoning. In view of the fact that it apparently can occur alone, I felt then that it deserved being placed in a separate category. For further discussion of its nature, see Scherer (1944).

(7) In so-called "natural cases of distemper," as diagnosed by the practitioner, which had died or were killed in the acute phase, even those showing some slight nervous manifestations, the brain showed no encephalitis, but might show some neuronal damage, perhaps a mild meningitis or glial clusters widely scattered in the brain. (Such glial clusters are illustrated in many medical textbooks, *e.g.*, Wertham and Wertham (1934) who state that they have been given an overestimated specific importance in veterinary neuropathology as a whole; it would appear that this may certainly have been so in distemper so far as any relation to "nervous symptoms" is concerned.)

(8) From canine cases of "encephalitis" which might, or might not have had a previous febrile background, distemper virus was never recovered by inoculation of brain material into ferrets although sometimes an infection, assumed to be distemper, resulted when ferrets were inoculated by spleen/blood from similar cases. (This coincides with later observations of MacIntyre *et al.*).

(9) Attempts made to transmit encephalitis from dog to dog have not been universally successful (Lalling, Bloom and others' private communications), which by itself was the strongest evidence that the distemper virus was not involved.

(10) Inoculation of distemper virus cisternally into dogs produced distemper but not an encephalitis (now also confirmed by the recent work of MacIntyre). On purely clinical grounds it was obviously impossible for anyone to make any inference on the role of the distemper virus in canine encephalitis. Pathological studies did not by themselves help to disentangle the problem because of the variety and severity of conditions found in what was said to be "*post-distemper encephalitis.*" Further, the pathological evidence indicated that the terms commonly used in a clinical sense, *e.g.*, fits, chorea, epileptiform encephalitis, meningitis, were vague and meaningless and could rarely be related to what was found pathologically; controlled experimentation was an obvious need. The few outstanding contributions up to 1948, require some attention because, if anything, they made matters more perplexing; the lessons learnt by Dunkin and Laidlaw had not been taken to heart.

5. Experimental Pathological Contributions until 1948

Posrednik (1931) described demyelination in natural cases of "distemper encephalitis" and produced similar lesions experimentally, but there is no indication of the source of his infective material; we certainly cannot be certain he was working with distemper. The same comment can apply to the work of Kantorowicz (1933), Puntoni (1923), and Robin & Vechiu (1926). The work of de Monbreun (1937) is also open to suspicion and deserves special comment. The second part of his paper deals with experimental *distemper encephalitis* and is full of excellent pathological description. The material from which subsequent inoculations were made was, however, obtained from a dog which died after *an acute illness of 30 hours* and this same hyperacute lethal disease was reproduced in experimental dogs. In neither pure distemper virus infections, nor in natural cases of distemper in dogs (if that can be diagnosed) is such an acute clinical syndrome seen; further, the ferrets reacted differently to infection as they did not develop coryza until the 25th day, an incubation period outside that generally accepted for distemper. The encephalitis produced in dogs also seemed to be of an acute haemorrhagic variety. Finally, the author describes and illustrates inclusion bodies in hepatic cells which are identical with those stated by Green to be due to fox encephalitis virus in dogs (which were also seen personally). There thus seemed some justification for Green's contention that de Monbreun had worked in fact, not with distemper, but with the fox virus; this is a noteworthy comment because de Monbreun's work has been largely quoted in connection with distemper pathology in general. This complexity is not made simpler by more recent important observations of Rubarth (1947); this author has written an extended work, of a standard rarely seen in veterinary pathology, of the results of an assiduous study of twelve years in Sweden. He describes a condition named "*Hepatitis contagiosa canis*" based upon 190 autopsies and extensive experimental work; it occurred mainly in young dogs (although up to four to five years was not rare) which showed a febrile illness of short duration and some symptoms quite like "distemper." The lesions were a dominant liver pathology with necrosis, fibrinous perihepatitis and oedema of the gall bladder, etc. Although some dogs showed paresthesia, blindness and convulsions, cerebral lesions were not prominent, but he mentions the presence of glial clusters and perivascular haemorrhages. It was proved to be an infectious and contagious disease, from which many dogs survived, caused by a filterable virus; dogs immune to this infection could be infected fatally with distemper. The interest of this work, as far as we are concerned, lies in the description of the universal presence of intranuclear inclusions in hepatic cells identical with those described by Green in fox encephalitis infection in dogs and by de Monbreun in his experimental dogs. Rubarth suggests that Green was in fact working with this virus of *Hepatitis contagiosa canis*! This disease has not been recognised so far in England* but it has been described in the States by Coffin (1948) and Riser (1948) while Bloom (private communication) has informed me that he recognised the condition ten years ago and now diagnoses it clinically; I have been privileged to see some of his pathological material. Kriesel (1938) also came to the conclusion that de Monbreun might have been working with the fox encephalitis virus and not with distemper; in his experimental dogs there was much dissimilarity in the course of the two infections; he noted the presence of the inclusions described by Green in 100 per cent. of the dogs infected with fox virus but they also were seen in 18 per cent. of the distemper virus infections in dogs! Cordy (1942) carried out some experimental work with dogs using a "commercial strain of distemper virus"; in one group he failed to produce lesions in the nervous system; it has been also mentioned that he failed to transmit "old dog encephalitis" (which bears a similarity to my lymphocytic perivascular encephalitis with diffuse sclerosis) to ferrets.

The other contribution of merit is that of Hurst, Cooke and Melvin (1943); the work of EWH on the encephalitides, neurotropic viruses and the demyelination problem is known to all workers in these fields. The paper contains an unrivalled account of pathological detail of demyelinating encephalopathy in dogs in Australia. He concluded that many of the lesions bore a close resemblance to those in the more severe demyelinating diseases of man. Bearing in mind what has been said about clinical and pathological diagnosis the same uncertainty might arise as to whether these were in fact due to the distemper virus. Hurst reproduced in experimental dogs identical demyelinating lesions, as seen in natural cases, with ease; the inoculating material was, however, obtained from his natural cases and he admits that some of the immunity tests were performed before initial infection had run its course. He fully discusses the question whether he was dealing with two viruses closely related if not antigenic duplicates. While this part of the work might be suspect, Hurst also produced the same lesion in dogs by intracerebral inoculation of authenticated distemper virus obtained from the Commonwealth Serum Laboratories. His claims therefore must be seriously considered but tempered by the more recent work of MacIntyre, Trevan and Montgomerie (1948).

In conclusion to this section it can be safely said that the claims based on experimental grounds regarding the role of the distemper virus in any variety of encephalitis, least of all a demyelinating form, rested on rather tenuous and controversial evidence. Most of the contributors on the positive side (except in some of Hurst's work) had derived their infecting inoculum from "natural street infections" and we know the difficulty attending absolute diagnosis of such cases. My own limited experimental work demonstrated clearly the many difficulties attached to work of this nature; further, that subsequent to pathological examination of many dogs we did not really know what we were trying to transmit from one case to the next. It was, however, clear that distemper virus could not be easily recovered from the brain from any type of encephalitis, and by us never at all. A demyelinating encephalopathy certainly has occurred in dogs for years and it seems as if this was due to some virus and that this virus might indeed produce an initial fever like distemper; it remained to be proven whether this virus was that of distemper as described by Dunkin and Laidlaw and which for generations has been said to be the major febrile scourge of dogs.

6. Neurotropic Viruses and Canine Encephalitis according to MacIntyre, Trevan and Montgomerie (1948)

This work was first communicated to the National Veterinary Congress in August, 1948; the authors describe five separate neuropathological entities in dogs. Their main conclusions are briefly enumerated from which it is clear that considerable advances have been made, many previous results can be better understood and some puzzling features of canine nervous disease explained

(*a*) *Distemper in dogs.* (Their Group Ia.)—Many cases of "*so-called encephalitis*" are associated with the invasion of the brain by the true distemper virus, yet the strains isolated from such

* No disease is, of course, "recognised" until it is found by looking with great care, and in many cases we must admit that autopsy work and selection of tissues for histological examination is done with much bias. We take refuge in saying that one cannot do everything! I have, however, seen cases of liver necrosis in dogs with a clinical history such as Rubarth describes, but I never looked for inclusion bodies in the liver!

cases showed no real neurotropic properties. They also observed that even when inoculated by the intracerebral route, typical distemper was produced and perhaps *without any signs* of nerve cell damage. Of the many thousands of susceptible dogs injected during the production of distemper virus less than 20 showed signs of nervous disease. In the brains of animals in this group which showed some signs of nervous involvement, neuronal damage, small haemorrhages were found but no inflammatory reaction and no demyelination. Ferrets inoculated with material from these cases behaved in the classical way; and strains of the virus were readily neutralised by anti-distemper serum.

(*b*) *"Hard pad disease"—with a demyelinating encephalopathy.* (Their Group Ib.)—This disease affected dogs of all ages which showed catarrhal symptoms, usually irregular and intermittent fever—sometimes a subnormal temperature persisted throughout the illness; severe diarrhoea was common. There might be some pneumonia but an outstanding lesion was a hyperkeratosis of the foot-pads appearing within one week of illness. At the same time nervous symptoms may develop—convulsions, screaming fits, blindness and paraplegia. The autopsy might reveal enteritis, cystitis, haemorrhage and oedema of the lungs and no macroscopic changes in the brain. Histologically the important lesions were the demyelinated foci in small scattered areas in cerebellar peduncles and folia, glial proliferation, swelling and proliferation of vascular endothelium, and intranuclear inclusion bodies in glia, histiocytes and ependyma. The injection into ferrets of tissue emulsions from foot-pads, spleen, brain or lung set up a disease like distemper but with an incubation rarely less than 17 days but often as long as 32 days and unlike distemper in ferrets, recovery was not uncommon. Attempts to transmit the disease to dogs produced only a febrile reaction lasting 14 days.

(*c*) *Demyelinating encephalitis.* (Their Group II.)—This form of disease was said to occur in dogs of all ages which showed mild catarrhal affections, dry cough with perhaps later development of nervous symptoms with convulsions, paralysis, chorea or ataxia, the animal dying in a coma. There were no lesions in the pads and the encephalitic lesions seemed to be like Group Ib. One prominent distinguishing feature was the complete inability to infect ferrets with Seitz-filtered material but in several attempts one dog injected intra-cerebrally became affected.

(*d*) (Their Group III).—Dogs in this group were said to develop an insidious nervous disorder without signs of generalised infection. The lesions here consisted of perivascular lymphocytic cuffing of vessels but no demyelination or neuronal damage. All attempts to transmit infection to ferrets or dogs failed.

Other types of encephalitis are briefly mentioned associated with toxoplasma (Group IV) and nutritional conditions (Group V) but they do not concern us here. It would seem, however, that at last some clarification has been reached regarding encephalitis in dogs and that we might discard the idea that the distemper virus under natural conditions, or experimentally, can produce an inflammatory, or a demyelinating form, of encephalitis. The difficulties of working with dogs to exclude distemper are clearly brought home; the previous confusion which arose on clinical grounds is apparent but some light may ultimately be shed on pathological entities, checked and rechecked with adequate experimentation. Out of chaos therefore we see some semblance of order. When more detailed pathological descriptions are available from this notable work of MacIntyre, Trevan and Montgomerie it may be possible to correlate some of the previous data of others. At the moment the type of encephalitis seen in either Groups Ib or II might coincide with the well-recognised, oft described, demyelinating lesion of other workers; on the other hand, Group Ib might be a new entity because of the peculiar hyperkeratosis of pads, while their Group II might represent what has so often before been called "post distemper encephalitis," my disseminated encephalitis and Scherer's "acute multiple sclerosis of dogs." (I have never at any time observed the pad lesions but this may not be surprising; I have mentioned in a footnote how blind pathologists can be and how lesions can be easily missed unless you are actually looking for them. The history of veterinary pathology is strewn with examples of this nature, for if more attention had been given in the past to rigorous examination for pathological detail many diseases would have been identified years before they actually were. That has been the result of an inordinate ardour for "microbe hunting"!) The lymphocytic encephalitis (their Group III) might correspond with my cases of diffuse sclerosis, with Cordy's "old dog encephalitis" and with the one case of Scherer and Collet. It would seem that this type is quite specific and if all are the same then it would appear to be non-transmissible to ferrets or other dogs.

Many problems associated with nervous disorders in dogs remain to be solved but many apparent complexities of the past have at least been partly clarified by the work of the Wellcome team. A virus has been shown to be associated with a demyelination lesion which by itself is most important, because the demyelinating diseases of man form probably the biggest problem confronting medical neurologists of to-day. Further experimental studies along the lines so admirably started may have many repercussions besides the immediate practical one of solving the causation of diseases of dogs and which is of importance to the veterinarian. For a long time it has been considered that viruses were not involved in the aetiology of any of the demyelinating encephalitides but this view must obviously be reconsidered. An immediate need would be the successful transmission of the disease from dog to dog but positive results by the use of brain emulsion should of necessity be tempered by the results of Kabat *et al.*, who have produced severe neurological manifestations and demyelination by the injection of heterologous and homologous brain emulsions in monkeys.

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Weekly Wisdom

Therefore Aristotle observes, "That the nature of everything is best seen in its smallest portions." Whence he seeks the nature of a commonwealth, first in a family; and so the nature of the world, and the policy thereof must be sought in mean relations and small portions. The magnetic virtue of iron was not first discovered in bars, but in needles.—Bacon's Novum Organum and Advancement of Learning. H. G. Bohn, London. 1853. p.81.

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ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Sheep Scab:

Denbighshire.—Tan-y-Coed Fields, Llanfair Talhavin, Abergele (January 27th).

* * * * *

Six cows, valued at between £400 and £500, were electrocuted at the farm of Mr. J. Paddock, Red House, Bicton, Shrewsbury. The theory is (says *The Meat Trades Journal*) that the cows must have had their noses in the water bowls, which were bolted to the tubular stalls and as soon as the hot-water boiler was switched on they were killed instantly.

CLINICAL COMMUNICATIONS

Canine Encephalitis

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After reading the paper on "Canine Encephalitis" by MacIntyre, Trevan and Montgomerie and the ensuing discussion in the issue of *The Veterinary Record* dated December 4th, 1948, I feel it may be of interest to quote from the case histories I have kept at the War Dogs Training Wing, R.A.V.C., in Germany, during the past 18 months.

In this school we breed our own puppies, almost exclusively Alsatians, for subsequent training as guard dogs. This branch of the school began in April, 1947, since when some 130 puppies have been born. Of these, a very large number have never reached the training kennels, due largely to two very severe outbreaks of encephalitis amongst them, the first occurring during February to March, 1948, as a result of which 20 of 34 infected puppies and adult dogs died or were destroyed, and the second between August and October, 1948, when 30 of 37 puppies infected, succumbed.

The two outbreaks were not dissimilar, but whereas the second definitely proved to be hard pad disease, there were certain features in the first which throw a doubt on such a diagnosis. On the other hand, the writer was almost entirely ignorant of the hard pad disease syndrome at the time of the first outbreak.

Outbreak No. 1

Symptoms

Those first observed in dogs inoculated against distemper by the serum/virus method (Behringwerke, Hannover, preparation) were inappetence, watery oculo/nasal discharge, listlessness and high fever, up to 105·4° F.

A tentative diagnosis of distemper was made, but since affected dogs had been inoculated against distemper at periods ranging between three weeks and one year previously, the diagnosis was highly unsatisfactory. It is inconceivable that so many inoculations, many of such long standing, could break down. Also the uninoculated puppies had repeatedly received prophylactic doses of anti-distemper serum before and therapeutic doses during the outbreak.

About ten days after the initial symptoms, the general picture presented was as follows: diarrhoea, general loss of condition, complete anorexia (in some cases), purulent ocular discharge, foul smelling brown slime on the teeth, necrosis and haemorrhage of the gums and in a few cases, also of the lips (no ulceration was evident in any case), blood-stained faeces, severe abdominal pain and respiratory distress, respirations being rapid and very laboured, temperature around 103° to 104° F. No vomiting occurred.

About seven days later, severe convulsions with frothing at the mouth and paddling motions with the legs occurred and some showed symptoms resembling meningitis. Others developed chorea and a few, dementia. These symptoms were followed by collapse and death, or the animals were destroyed.

The above symptoms were largely confined to puppies between the ages of four and a half to six months. (All immunised as from four months old.)

In ten dogs aged nine months to five years which became affected the symptoms were similar, but there were no convulsions. All recovered following treatment of a routine nature, with the exception of one which became severely dehydrated, collapsed and died.

In the case of eight uninoculated puppies aged two months which became infected early, symptoms were more or less identical except that there was no necrosis of the gums and the convulsions, which were invariably rapidly followed by collapse and death or destruction, were characterised exclusively by frothing at the mouth and champing of the jaws.

Post-mortem Findings

All animals which either died or were destroyed were subjected to *post-mortem* examination. Findings were similar in the majority of cases, and were as follows:—

Gastritis, enteritis, blood-stained faeces (in some cases), mild cystitis.

In some, there were varying degrees of pneumonia.

In those cases which showed symptoms of meningitis, congestion of the dura mater was demonstrable. In all other cases, macroscopical examination of the brain revealed nothing.

Owing to the difficulty of obtaining laboratory services at this time, only one brain was examined microscopically. The puppy (six months of age) had shown symptoms of dementia. The findings reported were: "Varying stages of degeneration in the ganglion cells."

Discussion and Conclusions

1. Early symptoms were suggestive of distemper but the facts that (*a*) so many inoculated dogs including adults became affected and (*b*) uninoculated puppies had received large prophylactic and therapeutic doses without results, tended strongly to rule out this diagnosis.

2. Later symptoms, especially the necrosis of the gums, were not consistent with either distemper or hard pad disease.

3. The one brain examined, however, gave findings indicative of distemper and not hard pad.

4. It would appear possible that this outbreak was an atypical distemper caused by mutation of the virus which, when it became more stable, possibly by virtue of animal passage in sub-clinical cases, produced at a later date the more characteristic syndrome described as Outbreak No. 2. This would tend to support the theory that distemper and hard pad disease, while obviously not a single entity, are distinctly related through a process of mutation of the virus.

Outbreak No. 2

About 60 per cent. of infected cases were immunised against distemper and in this outbreak the first cases were in these inoculated puppies. (The same preparation had been used as in the case of Outbreak No. 1.)

In-contacts again received very large prophylactic and therapeutic doses of anti-distemper serum but no advantageous results were observed.

The disease appeared to run one of three courses—peracute, acute and chronic.

1. Peracute

Symptoms

Sudden collapse with a blood-stained anal discharge and death. In cases where death did not occur for 48 hours, violent convulsions occurred. As the cases of sudden collapse and death were found in the morning, the possibility of convulsions having been present cannot be overlooked.

2. Acute

Symptoms

Serous oculo-nasal discharge, with a fluctuating fever ranging between 103° and 105·5° F., followed within ten days by hardening and thickening of the pads and in some cases by a similar change in the nose. There was enlargement of the submaxillary, prescapular and popliteal lymphatic glands. Diarrhoea was fairly constant, and often blood-stained. The appetite in some cases remained good, although others ate little. Breathing was often laboured, but in only four cases were pneumonic lesions demonstrable, and then not in any way in sufficient degree to warrant such respiratory distress. The laboured breathing, it was decided, was caused by abdominal pain which was a feature of many cases. Dehydration was a fairly common feature. Convulsions usually supervened within eight days of the onset of the pad lesions, followed in most cases by collapse and death.

Convulsions varied considerably in character:—

(*a*) The most typical form was champing of the jaws with foaming at the mouth, lasting anything from 30 seconds to four minutes, after which the dogs appeared much relieved and bright. Some cases lay down during the convulsions, but others remained sitting and just prior to the onset of an attack, a wild-eyed expression was noted. In the recumbent type, there was also a paddling movement of the legs, often very violent.

(*b*) Similar to the above, but prior to the convulsions, the dog would stand with a dazed expression, head hanging and often pressed against the kennel wall as in cases of meningitis.

(*c*) Choreic spasms of varying groups of muscles, followed by any of the above manifestations.

Urine and faeces were sometimes voided during spasms. Some cases became hyperaesthetic and attempted to bite when handled. A few tried to escape, and one which did, ran blindly about and headed for water.

3. Chronic

At the onset, the symptoms were similar to those seen in the subacute cases, but no symptoms of encephalitis appeared in the average period of seven days following thickening of the pads. There was apparent complete or partial recovery, but this was followed by choreic spasms of increasing violence in one case and by muscular inco-ordination in the other. This latter case has since made a complete recovery.

Post-mortem FINDINGS

These varied with the course of the disease.

Peracute cases showed varying combinations of the following: Mild catarrhal inflammation of the small bowel, often patchy in nature; haemorrhagic enteritis of the large intestine; uncoagulated blood or blood-stained exudate in the abdominal cavity; dark blood-stained oily fluid in the stomach and intestines; congestion and slight enlargement of the liver; small pneumonic foci on one or both lungs. In a few cases, no macroscopical changes were noted in the body cavities.

Of two cases sent for bacteriological examination, one was reported as having a very mild *Bact. coli* infection of the heart and spleen and the other proved negative.

More prolonged cases showed typical thickenings of the pads and in two cases also of the nose. Many of the above changes were also found. There were also oedema of the lungs and subpleural petechiae. Petechiae in the bladder and engorgement of the gall bladder were also noted.

Of two chronic cases encountered, one recovered after showing pad lesions and following a prolonged period of convalescence, but no convulsions, and the other was destroyed on the 35th day of illness after showing apparent complete recovery, which was followed by severe choreic spasms commencing on the 30th day. In these cases *post-mortem* examination revealed slight gastritis and severe patchy enteritis involving the whole intestine. There was a small quantity of blood in the intestinal lumen. All other organs appeared normal.

Since the nature of the disease has been determined, no brains were subjected to microscopical examination.

TREATMENT

In the early stages of the outbreak the exact nature of the disease was not recognised, and symptomatic treatment was adopted. Sulphanilamide and sulphathiazole were used to combat catarrhal symptoms and fever. This line of treatment was discontinued when it was realised that the condition was hard pad disease.

Admittedly no improvement was noted following the administration of these drugs, but it is impossible to say whether their administration had a direct bearing on the mortality rate or not. However, five six-months-old dogs thus treated, and which received no other medicinal treatment, did recover after showing serous oculonasal discharge and fever ranging from 103° to 105° F. It is possible that these cases were not hard pad disease, although it seems unlikely, since they were direct contacts with cases which developed typical symptoms and which died or were destroyed. One of these five puppies showed mild convulsions about a week after apparent recovery, but these did not recur.

Other symptomatic treatment to combat the fever, diarrhoea, abdominal pain, etc., did little to influence the course of the disease.

Small quantities of liver extract (Pernacol forte) and of injectable vitamin B_1 (Betaxin) were available and it is interesting to note that both the cases which received these preparations intramuscularly in doses of 2 c.c. on two consecutive days, repeated in six days, and 1 c.c. daily for six days respectively, became chronic. In the latter case, nicotinic acid was also administered *per os*.

No specific anti-serum was available.

Diet.—In the case of puppies involved in both outbreaks, wherever possible the diets were reinforced with vitamins and mineral supplements, *e.g.*, cod liver oil, syr. ferri phos. co. in the proportion of 2:1, mag. sulph, sodium chloride, vitamin B and vitamin C, etc. In certain cases Vigantol was administered by subcutaneous injection or *per os*. The diet itself, as far as was practicable, consisted of fresh meat, liver, milk (powdered) and small quantities only of biscuits. Green vegetables were given, and were preferred raw almost without exception.

It is worthy of note that of eight uninoculated puppies of the same litter aged four months, all of which were in direct contact with cases which developed typical symptoms, only three showed atypical symptoms. Two of these died, and the third made an uneventful recovery. The remainder were normal and apparently healthy throughout. Whether this was due to hybrid vigour or not cannot be said, but the fact remains that despite their having every opportunity to contract the disease, they remained healthy during the whole outbreak.

CONCLUSIONS

1. In this outbreak it appears that dogs immunised against distemper were in no way less susceptible to hard pad disease, whereas five uninoculated mongrel puppies which were in direct contact with infected cases, themselves showed no symptoms of disease.

2. Young dogs up to six months of age were alone susceptible, for whereas isolation of affected cases was carried out as efficiently as possible, at no time was this complete. This was due to the close proximity of the whelping pens and puppy runs to the Main Kennel blocks and to the Surgery and Hospital premises.

The intermixing of handlers after working hours must also have accounted for the spreading of infection from the puppy pens to other kennels. No adult dogs, including nursing bitches one of whose puppies died of the disease, however, became clinically infected.

3. Liver extract and vitamin B_1 intramuscularly appeared, in the two cases in which they were used, to have some effect in delaying, if not altogether obviating, nervous symptoms.

SUMMARY OF CASES

Age. * Denotes inoculated against distemper by the serum/virus method	Admitted	Died	Destroyed	Cured
* Nine months to 5 years	10	—	1	9
* Four to 6 months ..	16	5	6	5
Two months	8	1	7	Nil
TOTAL	34	6	14	14
		(2)		

SUMMARY OF CASES

	Inoculated	Uninoculated	Unweaned	Total
Total puppies in school ..	23	17	28	68
Total affected	23	12	2	37
Total unaffected	Nil	5	26	31
Total died or destroyed ..	17	11	2	30
Total recovered	6	1	Nil	7

Note.—Of the unweaned puppies which died, only two showed symptoms or *post-mortem* findings which could point with any certainty to hard pad disease. Only these are shown as infected in the summary, deaths occurring on the day of birth or during the first three days of life being discounted (*i.e.*, a further seven).

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Hard Pad Disease

A. NOEL ORMROD, M.R.C.V.S.

WOODCHURCH, WIRRAL

In my practice the condition which we now know as hard pad disease has become a major problem in the last few years. I am convinced that many cases remain undiagnosed and that failure on the part of the profession to detect the early cases, coupled with complete ignorance of the disease on that of the dog-owning public, are responsible for the gradual but positive development of the epidemic. In the hope of provoking discussion and gleaning further enlightenment from others more capable than I, I venture to publish my own observations on this particularly troublesome disorder.

This disease mainly affects young animals but cases have been observed in dogs of four and six years old: I have not seen a case in puppies of under five months. It is characterised by its insidious onset and the case is often well advanced before advice is sought. The earliest symptoms are diarrhoea, which is often missed by an unobservant owner, and vomiting, which, although often troublesome, is rarely very distressing to the dog. In fact, the patient is frequently bright and cheerful in spite of the vomiting and the temperature, which is found at this time to be around 103° F. The nose remains cold but excessively moist, with a characteristic "dewdrop" between the nostrils. The appetite may be completely or only somewhat in abeyance. If diarrhoea and emesis are treated at this stage there is usually an apparent rapid improvement and if seen again after three or four days the owner is usually pleased with results. When the temperature is taken, however, it is found to be still around 103° F. Enquiry elicits the information that the appetite, whilst improved, has not returned to normal. The pads are seen to be becoming hard and smooth, the hardness being most easily seen at the periphery of the pad which now has a sharply defined edge. From this stage on, the pads continue to harden and thicken and may become highly polished. By this time the nostrils are seen to be becoming hard and crusted with a sticky adherent scab. A positive diagnosis is now easy.

It is frequently difficult to convince the owner in the face of the apparent improvement and the dog's good spirits, that the condition is serious. At this stage it has been my practice, lest the sceptical owner should think that I was "running up an account" unnecessarily, to leave my next visit provisionally for seven to ten days, but to give instructions for treatment to proceed and great care to continue to be taken of the patient; also that, should any

"nervous" symptoms occur before the next visit is due, I should be informed. Frequently, to my regret, I have been recalled after a further few days, to find that a young, bright intelligent animal has deteriorated into a palsied idiot. The pads are now very hard, either highly polished or cracking. The appetite is either completely absent or voracious, in which case the dog is seen to be eating in a typically frenzied manner. Co-ordination of movement is grossly disturbed, vision is impaired and the animal frequently wanders aimlessly about the room. There may be whining or a quiet whimpering; in short, the classical symptoms of a severe encephalitis are present.

Cases where typical encephalitic symptoms have developed have been treated without success.

Many cases which have been diagnosed early, *i.e.*, before or during the quiescent phase that occurs between the primary stages of gastro-intestinal disturbance and the final stage of encephalitis, and when the only indications of the presence of the disease are the elevated temperature, hard pads and history of earlier symptoms, have been treated successfully with massive doses of Pyridoxin (20 to 40 mgm. *t.i.d.*) supported by symptomatic treatment of the gastro-intestinal disturbance and the use, in certain cases, of a simple sedative such as bromide and chloral.

It was noted quite early that sulphonamides were contra-indicated, as also are barbiturates and Prominal. (I might mention here, however, that the first case which was seen by me, in 1940, was treated successfully by the rather heroic use of Prontosil rubrum. I am now inclined to believe that this patient recovered in spite of treatment, which was countered by particularly devoted and skilful nursing by the owner.) The cost of Pyridoxin, which is from 9d. to 1s. per 20 mgm. tablet, is a factor which has to be borne in mind.

Cases in young animals of five to nine months and those in a debilitated state have all been fatal.

I do not consider that there is any breed susceptibility, but I have seen more cases in sheepdogs than in any other breed.

ABSTRACTS

[The Effect of Certain Chemical Agents on the Virus of Newcastle Disease of Chickens. Cunningham, C. H. (1948.) *Amer. J. vet. Res.* **9.** 195.]

A number of chemical agents have been tested against a strain of Newcastle disease virus. The strain used had been cultivated through 15 passages in the allantoic sac of chick embryos, and was known to be lethal within two days to these embryos.

In the tests one part of virus suspension (allantoic fluid) was added to nine parts of test solution and allowed to stand at room temperature for three minutes before injection of 0·05 ml. (*i.e.*, a calculated 10^5 embryo M.L.D.s) into the allantois of ten to 12 day embryos. Survival of the embryos for six days subsequent to injection was considered evidence that the virus was inactivated. Controls were injected with test solution alone.

Eighteen substances were tried, and a number found to be effective. For further details the original article should be consulted.

J. G. C.

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[The Efficacy of Methyl Bromide as a Fumigant for the Prevention of Caecal Coccidiosis in Chickens. Boney, W. A. (1948.) *Amer. J. vet. Res.* **9.** 210.]

The efficiency of methyl bromide as an inactivator of sporulated oocysts of *Eimeria tenella* was tested under conditions closely simulating those found on the average poultry farm.

The rate of contamination was eight to 16 million infective oocysts per sq. foot of litter (soil, cane pulp on soil, and cane pulp on wooden floors). Methyl bromide was applied to some of the infected areas, others being untreated to serve as controls.

It was found that 1 lb. of methyl bromide per 1 000 sq. ft. effectively inactivated the sporulated oocysts, thus demonstrating the possibility of using this fumigant as an economic method for the control of caecal coccidiosis.

J. G. C.

* * * * *

[A Note on the Collection of Live Adults of *Trichinella spiralis.* Ross, W. M. (1948.) *Canad. J. comp. Med.* **12.** 152-154.]

Trichinella larvae were easily separated and collected in a modified Baermann apparatus: the collection of living adults in large numbers was more difficult but was effected as follows.

Rats are infected with larvae given in saline and killed five days later. They are given only water for the 24 hours before death. The gut from duodenum to caecum is removed immediately after death and opened in a beaker of warm saline. Any gut contents are removed by gentle rinsing in the beaker. The gut is then transferred to a shallow pan containing warm saline, and the mucosa removed completely by using the narrow end of a glass slide as a cutting tool; the muscular portion of the gut is then discarded after rinsing in saline. Mucosal shreds can be finely divided by rubbing them between the fingers and against the sides of the pan; when this is accomplished, the gut washings in the beaker are added to the contents of the pan.

The suspension of worms and mucosa in saline is then flooded on to a piece of filter paper by means of a pipette with bulb attached. The filter paper is placed on top of several layers of blotting paper, or even newspaper, and in this way the excess saline is removed. Whatman Filter paper No. 2 seems to be about the best for this purpose; a coarser quality paper will absorb the moisture more readily but, at the same time, will introduce fibres which are difficult to separate out later.

The filter paper is then turned over and placed face down in a petri or culture dish containing warm saline; the adult trichinae, stimulated by the heat, detach themselves from the paper and fall to the bottom of the dish, leaving the mucosal shreds behind. Undigested food particles, however, will not adhere to the paper and will present a definite problem in obtaining the worms free from debris if the animal is not starved before killing. During collection of the adults the petri dish must be kept warm and this is best done by placing it in the sink in flowing hot water.

The adult trichinae, now concentrated in a small volume of saline, are quite free from mucosal shreds or fibres. If necessary this concentrate itself may be flooded on to a fresh piece of filter paper to remove any remaining debris, and the worms again collected in a smaller petri dish.

It is most important to keep the saline warm during the entire procedure; temperatures up to 42° C. have been used without any indication of intolerance on the part of the adults. At lower temperatures the worms are inert and will not detach themselves from the filter paper. They also appear to be quite lethargic in distilled water, even at suitable temperatures.

J. S. S.

REVIEW

[I.—Phenothiazine (Thiodiphenylamine) in Veterinary Practice: A Review of the Literature of 1942-46. By J. Tweedale Edwards, M.R.C.V.S.; **II.—Bibliography of Phenothiazine as an Anthelmintic, 1942-46.** By The Imperial Bureau of Agricultural Parasitology (Helminthology).]

In 1942 Davey and Innes published a review of the early development of phenothiazine therapy in animals and men (*Vet. Bull.* **12.** pp.R7-R14). Mr. Edwards has now taken up the story to the end of 1946 and is to be congratulated on a very interesting and comprehensive review which will be of great value, not only to research workers, but to all who are concerned with the clinical application of this well-known drug.

After preliminary sections on Pharmacology and Toxicology, under which headings comparatively little progress has been made since 1942, the author devotes the major portion of his review to the detailed description of the use of phenothiazine in treatment or prevention of helminthiasis in domestic livestock. He sets out fairly and without bias the various views put forward by authorities who have published work on the drug and provides, in each section, a useful table summarising the action of phenothiazine on the various worms infesting a particular host. In the section devoted to sheep the lungworm is noted as "*Dictyocaulus viviparus*"—a species which is found only in cattle and deer—but this error is really of only minor significance.

In relation to the use of phenothiazine for the control of helminths in sheep the dosage recommended by various authorities shows considerable variation, but this must be considered in the light of the observations of Gordon (1943), who determined that while *Haemonchus* can be removed by a very small dose (6 to 10 g. for an adult sheep) the other stomach worms require more and the intestinal worms still higher doses. In the writer's experience disappointment with the results of prophylactic dosing in sheep often results from under-dosage, particularly where intestinal trichostrongyles are mainly concerned.

This publication includes a bibliography of phenothiazine as an anthelmintic, prepared by the Imperial Bureau of Agricultural Parasitology (Helminthology). This omits a number of publications which have appeared during the years under review but, in the main, the subject matter of the papers omitted is well covered by others which are included.

The bibliography will be particularly valuable to research workers.

Questions and Answers

The submission of questions for inclusion in this column will be welcomed: they can relate to any aspect of veterinary work. For purposes of record, each question is numbered, and those submitting questions are required to furnish (not for publication) name and address.

Answers to readers' queries represent exclusively the personal opinions of the writers, and criticism of the replies will be appreciated.

All communications should be addressed to the Editor.

Imperforate Teats

Q. CCLXXXV.—*Would you consider imperforate teats in heifers congenital or due to some hereditary factor in the sire?*

[*The following case will elucidate my question:—*

A pedigree Ayrshire bull was loaned to another Ayrshire breeder. This bull produced six heifer calves, each of which possessed one imperforate teat (either near or off fore), each visible several months before these heifers had their first calf, being considerably larger than the remaining three teats.

The teat duct was completely blocked from top to bottom, except for a teat valve at the tip of the teat, with perhaps an inch only of duct.

There had been no previous history of imperforate teats in this herd, until the introduction of this one bull. Other progeny of this bull had up to this time been normal.]

A.—The wording of this question clearly implies that there is a confusion in the mind of its poser in regard to correct technical usage of the terms "congenital" and "hereditary." The matter of their definition need not, however, concern us here, for it is quite clear from the accompanying history that the questioner simply wants to know whether or not the imperforate teat condition which he describes is attributable to an hereditary cause.

The occurrence of an identical teat defect in each of six heifers by the same bull must certainly be regarded as strong evidence that the abnormality is genetic in origin. It is impossible to define accurately the mode of inheritance for the defect owing to the lack of essential data in the history of the case provided by the sender of the question. On the basis of recorded observations on similar genetic phenomena in animals and man, the probability is that the abnormality is due to a mutant gene which is transmitted from one generation to the next in recessive Mendelian fashion. The incidence in the manifestation of abnormality would be only slightly complicated by the fact that the expression of the mutant (or abnormal) character is obviously sex-limited

The assumption that the gene concerned with the defective teat structure is most likely to be recessive is in full accord with the general genetical truism that a given normal gene usually tends to be dominant to its mutant allele.

. . . . . .

Recurrent Sores on Cows' Teats

Q. CCLXXXVI.—*What is the cause of, and what treatment is advised for, a firm circular running sore about the size of a half-crown met with on cows' teats? It usually persists throughout the summer months, returning each year, but not always on the same teat.*

(I do not refer to the small cow-pox sore or the sore on the udder between the front teats.)

A.—This condition is not infrequently seen in cows following upon a sore on the teats, from any cause, which becomes infected and irritable, particularly in the summer when flies are prevalent. There is no doubt the cow licks the sore on the teat herself, thus increasing its size. This licking is a habit which can only be overcome by keeping the cow in and tied up so that she cannot lick the sore, and thus allowing it time to heal.

. . . . . .

Infertility in Hornless Billy Goat

Q. CCLXXXVII.—*An eight-months-old hornless British Saanen billy goat is proving infertile with proven dams. Both his parents are hornless B.S.*

The breeder wishes to have an authoritative opinion as to whether this infertility could be due to the fact that BOTH *parents are hornless. In that case presumably the condition would be permanent.*

If not, could any treatment be suggested and how long would it be before results were evident?

A.—For many years it has been the practice, among goat breeders, to breed from hornless parents with the express purpose of breeding out the horn and producing a hornless goat, and until recently only hornless billy goats were accepted for registration. The infertility of this eight-months-old hornless B.S. billy goat cannot be explained merely by the fact that both his parents are hornless, because it is well known that fertile sires are produced by hornless parents, although, no doubt, on the other hand, infertile ones have also been bred. If he is not one of the latter some other cause of his infertility should be sought. There is a school of thought that believes that the effort to produce a hornless strain is causing the production of a larger number of hermaphrodites, this being the reason why so many were anxious for the reintroduction of the horn. It is quite impossible to suggest treatment as no diagnosis is arrived at, but it should be borne in mind how irregularly some individual goats react to oestrogens. If this animal is hermaphroditic the condition will be permanent, but if his genitals are normal an examination of his sperm will give some indication of the line of treatment to follow. At his age an improved diet might assist and he should not be used oftener than at weekly or longer intervals.

. . . . . .

Coat Eating in Dogs

Q. CCLXXXVIII.—*Opinion is sought as to whether a condition in which dogs eat their own coats is a vice comparable with coprophagy, dealt with in your reply to Question CCLX.*

A.—The respondent is not aware of eating of the coat in the dog occurring as a vice.

The coat is pulled out and some of it may be swallowed when biting during the course of irritant skin diseases or where the coat has become matted in cases of neglect.

There also appears to be a definite tendency in bitches to pull out the hair from the flanks shortly after whelping, or associated with pseudopregnancy.

* * * * *

Correspondence

Q. 281: "Certificate for Horse with 'Technical' Unsoundness"

Sir,—I read the answer to this question with great interest though I do not agree that the word sound should be avoided.

Your respondent did not answer the second part of the question. It is awkwardly worded, as there is no such thing as technical unsoundness. Soundness is a matter of opinion and fortunately most veterinary surgeons are agreed as to the significance of the various conditions met with. The fun starts when two or more of them disagree.

The case put forward in the question can be met by finishing the certificate as follows:—

> I observe that the gelding described above has a splint on the inside of each foreleg. I do not consider that these affect or are likely to affect his usefulness.
>
> This animal is, in my opinion, sound.

Yours faithfully,

Lambourn. A. C. Fraser.

January 30th, 1949.

. . . . . .

Umbilical Hernia in the Foal

Sir,—I do not wish to be dogmatic or disparaging, but I regard the use of a clamp for treating this condition as out of date and consider that it should be relegated to the limbo of the past, in the same way as the vulvar clamp and the "girdle of chastity."

In simple cases, the application of a cantharides blister will often bring about a cure. Good results have been claimed by injecting white paraffin close to the hernial opening (*i.e.*, Gersuny's wax operation, as devised for prolapsus recti in the dog). A few years ago a medical year book mentioned the use of an acid injection in cases in the human subject.

But to those endowed with the gift of surgery the radical operation devised by Mayo will no doubt appeal.

Yours faithfully,

Winchester. J. F. D. Tutt.

January 31st, 1949.

If a milk producer gets only from £20 to £30 for a cast cow, and he knows, on the other hand, that he can get from £80 to £100 for a good heifer, he will be tempted to part with the heifer rather than use her for herd-replacement. In that event he can maintain his herd numbers only by keeping some cows that otherwise should be cast. Hence, unless these matters can be better balanced, there is the danger (the South-West figures suggest that the danger has already been actualised) of many milk producers being led into habits of bad dairy husbandry. What is the solution? Is there a ready means of redressing the balance? *Farming News* (Glasgow).

QUESTIONS IN PARLIAMENT

Horseflesh (Price)

Mr. G. Williams (Jan. 31st) asked the Minister of Food if he will consider reducing the controlled price of horseflesh in order to discourage the slaughtering of sound working horses.

Dr. Summerskill: No. I doubt if such action would have the desired effect.

Horses (Export)

Mr. G. Williams (Jan. 31st) asked the Minister of Agriculture whether he is aware that a substantial number of farm horses are being exported from this country for slaughter in Belgium and Holland although they are young and fully capable of work; whether he is satisfied that there is a surplus of horses in this country; and whether he proposes to take any action in the matter.

Mr. T. Williams: I would refer the hon. Member to my reply to my hon. Friend the Member for Acton (Mr. Sparks) on January 24th. In addition to the figures there given for Belgium, an average of about 26 horses per week went to Holland in the last quarter of 1948, and a total of eight in the preceding nine months. Licences for the export of horses are only issued within limits which are settled from time to time after consultation with the Farmers' Unions of Great Britain and are designed to ensure that the requirements of British agriculture are not prejudiced.

Sir R. Glyn asked the Minister of Agriculture whether his attention has been drawn to the increasing number of British working horses exported to the Continent for food; and whether he is satisfied that this drain on our best horses is in the interest of British agriculture in time of emergency when fuel supplies may be difficult to obtain for tractors, etc.

Mr. T. Williams: I would refer the hon. Member to the reply that I have given to-day to a Question by the hon. and gallant Member for Tonbridge (Mr. G. Williams).

Sir R. Glyn (on February 3rd) asked the President of the Board of Trade whether his Department are required to provide licences for persons desiring to export shiploads of working horses, bred and owned in the United Kingdom, to Belgium and other Continental ports; and what are the number of such animals leaving British east coast ports each week.

Mr. H. Wilson: The export of all horses is subject to licence. During the six months ended December 31st, 1948, 62 licences were issued for the export of 2,897 working horses to the Continent, but I cannot say how many of these have been actually exported, since such exports are not separately recorded in the trade returns.

NOTES AND NEWS

Diary of Events

Feb. 12th.—Meeting of the Biochemical Society at the Westminster Hospital Medical School, 17, Horseferry Road, London, S.W.1, 11 a.m.

Feb. 16th.—Meeting of the Section of Comparative Medicine, Royal Society of Medicine, 1, Wimpole Street, W.1, 5 p.m.

Feb. 18th.—Meeting of the Sussex Division, N.V.M.A., at Brighton (Old Ship Hotel), 2.30 p.m.

Feb. 18th.—Annual meeting of the North of Ireland Division, N.V.M.A., at The Farm, Stormont, 2.30 p.m.

Feb. 23rd.—Annual Meeting of the South Eastern Division, N.V.M.A., at Maidstone (Royal Star Hotel), 2.30 p.m.

Feb. 23rd.—Annual Meeting of the Scottish Metropolitan Division, N.V.M.A., at the Royal (Dick) Veterinary College, Edinburgh, 2.30 p.m.

Feb. 24th.—Meeting of the Dumfries and Galloway Division, N.V.M.A., at Carlisle (Crown and Mitre Hotel), 2 p.m.

Feb. 24th.—Glasgow Veterinary College Annual Ball, at Cranston's, Royal Exchange Square, 8 p.m.

Feb. 25th.—Annual Meeting of the Royal Counties Division, N.V.M.A., at Reading (Caversham Bridge Hotel), 2.30 p.m.

Feb. 25th.—Liverpool University Veterinary Society Annual Ball.

Feb. 26th.—Meeting of the Society of Women Veterinary Surgeons at the Royal Veterinary College, N.W.1, 2 p.m.

Mar. 9th.—Meeting of the Southern Counties Division, N.V.M.A., at Bournemouth, 6 p.m.

Apr. 5th, 6th and 7th.—Quarterly Meetings of Council, R.C.V.S.

Aug. 8th to 13th.—Fourteenth International Veterinary Congress, Central Hall, Westminster, and Church House, Westminster, London, S.W.1.

N.V.M.A. Membership Subscription

The attention of members is drawn to the following Article of Association of the N.V.M.A.:—

> 35. During the first week of March in each year, notice shall be sent to all members whose subscriptions shall be unpaid. Should the subscription of any member remain unpaid by the 30th day of June, he shall cease to be entitled to receive the publications of the Association or to attend or vote at any General Meeting but shall remain legally liable for his subscription. If the subscription of a member shall be in arrear for one year he shall cease to be a member of the Association.

In the past, through failure to pay their subscriptions when due, the Association has been in the unfortunate position of having to remove the names of a number of members when it had been only a matter of inadvertence that the subscription had not been paid. The Officers of the Association will be pleased if this position can be avoided in future by members recognising the need to have their subscriptions paid in good time.

The majority of members find a banker's order the most convenient method of payment, and the General Secretary will be glad to send one of these forms to any member desirous of adopting this system.

* * * * *

R.C.V.S. Luncheon to Minister of Agriculture and Fisheries

The President of the Royal College of Veterinary Surgeons, Mr. H. W. Dawes, gave a small and informal Lunch Party for the Minister of Agriculture, Mr. Tom Williams, M.P., with whom were his Parliamentary Secretaries, the Earl of Huntingdon and Mr. G. A. Brown, M.P., and the Permanent Secretary, Sir Donald Vandepeer. Mr. H. J. Johns and Mr. McGilvray were also present.

The President was supported by Professor J. B. Buxton, Treasurer of the R.C.V.S., and Professor T. Dalling, Vice-President of the R.C.V.S., who was also present in his capacity as Chief Veterinary Officer.

Also asked to meet the Minister and his staff were the President of the National Veterinary Medical Association, Dr. Montgomerie, and the immediate past-President of the Association, Mr. L. Guy Anderson, who held office during the passage into law of the Veterinary Surgeons Act, 1948. Veterinary representatives of the Loveday and Chancellor Committees present were respectively, Dr. W. R. Wooldridge and Lt.-Col. P. J. Simpson.

There were no formal speeches but after Mr. Dawes had welcomed the guests both from the Ministry and from the veterinary profession, Mr. Tom Williams replied briefly but in a happily phrased speech.

The cordial atmosphere of the small gathering was felt by the veterinary representatives and was endorsed by the Minister's good wishes and remarks regarding the future of the veterinary profession.

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R.C.V.S. REGISTER REMOVALS

By order of the Royal College of Veterinary Surgeons, the names of George Leo Bradley, 160, Watling Street, Towcester, Northants, formerly of 96, Crofton Park, Yeovil, and Finlay George Fraser, 56, Newgate Street, Morpeth, Northumberland, formerly of Cust Cottage, Appleby, Westmorland, have been removed from the Statutory Register of Veterinary Surgeons.

* * * * *

FIFTH INTERNATIONAL CONGRESS OF COMPARATIVE PATHOLOGY

The Council of the Royal College of Veterinary Surgeons has appointed Mr. R. E. Glover, who is Honorary Secretary of the British National Committee of the above Congress, to be the official representative of the Royal College at the Congress.

* * * * *

Dishorning by Means of Rubber Bands

As mentioned in an earlier issue, the Officers of the Association sought advice as to the legality of this method of dishorning. The Ministry of Agriculture has now confirmed the view which we recently expressed, in the following words:—

> "The Animals (Anaesthetics) Act, 1919, as amended by the Order of September 13th, 1948, makes it illegal to perform the operation of dishorning cattle over one month old, unless during the whole of the operation they are under the influence of some general or local anaesthetic of sufficient power to prevent them feeling pain.
>
> "In the first instance, it is understood that the statement that the rubber band method is painless is not universally accepted

and, secondly, some doubt has been raised as to whether, in the narrow sense of the word in which it is used in the Act of 1919, the method constitutes an operation. It is obvious that there is scope for legal argument here. Does the Act, when it refers to operation, cover such a method?

"The Ministry is, of course, unable to express any authoritative opinion on this matter: that can only be done by Courts of Law. That being so, it is clear that veterinary surgeons who use the method can be prosecuted, though there may be little likelihood of that happening."

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PERSONAL

Births.—Nixon.—On January 27th, 1949, to Mary (*née* Phillips) and George Nixon, 1, Gainsborough Road, Bury St. Edmunds—a brother for Robert.

Roberton.—At Kingsfield House, Faversham, Kent, on January 6th, 1949, to Agnes (*née* Menzies), wife of David P. Roberton, M.R.C.V.S., a daughter—both well.

Wood.—On January 11th, 1949, to Jean, wife of J. C. Wood, of 1, Church Lane, Stoughton, Leics.—a son, Michael Anthony.

* * * * *

Forthcoming Marriages.—Cabot—Blampied.—The engagement is announced between Sir Daniel A. E. Cabot (late Chief Veterinary Officer, Ministry of Agriculture and Fisheries), Ommaroo Hotel, Jersey, and Anne le Cornu Blampied, of L'Abri, La Rocque, Jersey.

Rogers—Smith.—The engagement is announced between Michael John Hugh Rogers, M.R.C.V.S., son of Mr. and Mrs. J. Rogers, Little Kimble, Aylesbury, and Audrey Georgina Smith, M.R.C.V.S., daughter of Mr. and Mrs. George Smith, Hill End, Upton-on-Severn.

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INTERNATIONAL SCIENTIFIC COMMITTEE FOR TRYPANOSOMIASIS RESEARCH

The above committee, which was formed as a result of the international conference of 40 scientists held at Brazzaville in French Equatorial Africa last February, held its first meeting in London during the past week. It is a non-governmental standing committee comprising scientists from the United Kingdom and colonies, France, Belgium, Portugal, South Africa, Southern Rhodesia, and the Sudan. The nucleus of the committee is the British Colonial Office tsetse committee, which finds the permanent secretariat.

Apart from representing different countries, the delegates also represent the three branches of tsetse research, medical, veterinary, and entomological, whose co-ordination is so necessary to the proper combating of the fly and the disease.

The conference heard reviews of the work in hand in the various countries of Africa. South Africa, for instance, has had great success in exterminating the fly by the use of D.D.T. smoke put down from the air. Southern Rhodesia has found destruction of wild game in selected areas effective. East Africa has concentrated on the classification of trypanosomes, while in West Africa they are still preoccupied with the disease in humans, now only a minor menace in the East. The Sudan has played an important part in the antrycide experiments.

The following attended:—

Members.—Professor J. Rodhain and Dr. L. Tobback (Belgium); Professor Roubaud and Colonel Sa'eun (France); Dr. Fernando de Cruz Ferreira (Portugal); Dr. G. R. Ross and Mr. J. K. Chorley (S. Rhodesia); Mr. J. T. R. Evans (Sudan); Dr. Rene du Toit and Dr. H. S. Gear (S. Africa); Professor P. A. Buxton and W. H. Potts, and Dr. J. Carmichael (U.K.)

Advisers.—M. Pierre Leboeuf (Belgium); M. Ballay (France); Mr. D. A. Lawrence (S. Rhodesia); Dr. E. M. Lourie, Mr. R. J. Simmons, Dr. H. M. O. Lester, Mr. W. H. Potts, and Dr. J. Carmichael (U.K.)

Observers.—Mr. R. McChery (S. Rhodesia); Dr. Mahaffy and Dr. Buchanan (U.K.). The Secretary is Mr. J. K. Thompson (U.K.).

* * * * *

ANIMAL HEALTH TRUST HOSPITAL CENTRES

Hampshire Moves

The County of Hampshire, which showed such initiative in the matter of the introduction of artificial insemination of cattle, again is taking the lead—in the Animal Health Trust's scheme for the establishment of a veterinary hospital in every county.

At a recent Press conference at Winchester, Sir Cleveland Fyfe (organising secretary of the Trust) said that no decision had yet been made regarding the site of the hospital for Hampshire, but the Trust had in mind the general outline of the work the hospital would do. It would be staffed by experienced veterinary officers, supplemented by trained staff. Until the hospitals were in being, the diagnostic service would be carried out at research stations, some of which had already been set up. The veterinary profession was behind the Trust in this matter.

"We have been in touch with the National Veterinary Medical Association, which is whole-heartedly in favour of the hospitals scheme," Sir Cleveland explained, adding that the hospitals would be run by veterinary surgeons themselves acting through the county's committee set up under the Animal Health Trust.

Sir Reginald Dorman-Smith, who is chairman of the Hampshire County Committee, said he was confident that warm support for the work the Trust was doing in promoting animal health would be given by every part of the county. Hampshire had been asked to raise not less than £3,000 this year, he said.

The Lord-Lieutenant of Hampshire (Lord Portal) is president of the Hampshire committee, which comprises, besides Sir Reginald Dorman-Smith, Mrs. F. Firbank and Mr. H. A. Lanning (Canine Societies), Miss Larkworthy and Mr. N. Cheke (poultry breeders), Messrs. F. J. Lerwill, J. Fawcett, J. Hedges (National Farmers' Union), Mr. A. F. G. Fletcher (N.U.A.W.), Mr. N. V. Andrews (foodstuffs firms), Mr. R. P. Chester (National Pig Breeders Association), Mr. G. N. Gould, and Mr. J. F. D. Tutt (veterinary surgeons). Commander S. K. Horne, R.N., is the county organiser and secretary.

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LEGAL NOTES

Order for Destruction of Dog: Justices' Jurisdiction Challenged.—*The Times* reports that on Tuesday of last week Mr. A. S. Orr applied to a Divisional Court—Mr. Justice Humphreys and Mr. Justice Finnemore—on behalf of Shirley Dodders, aged 11, of Penzance, for leave to apply for an order of *certiorari* against Penzance justices.

Mr. Orr said that the applicant was seeking to quash the order for the destruction of her dog made by the justices on December 17th last. In August a summons was issued under section 2 of the Dogs Act, 1871, and the applicant was ordered to keep the dog under proper control. In December a further summons was issued under section 34 of the Summary Jurisdiction Act, 1897, alleging that she had failed to comply with the provisions of the order. The justices accordingly ordered the destruction of the dog.

Mr. Orr contended that the justices had no jurisdiction under the Act of 1897 to make such an order, which could only be made under the Dogs Act.

Mr. Justice Humphreys said that there was certainly something to argue, and leave would be given.

* * * * *

RECORD MILK OUTPUT

Mr. Williams, Minister of Agriculture and Fisheries, speaking at Dorchester on Saturday of last week, said it was heartening to see that milk production had been expanding continuously. Milk sold over the whole year was a record, with an increase of 144 million gallons on 1947. Progress had been due rather to an increase in yields than to an increase in the number of dairy cows.

* * * * *

To improve the quality of whalemeat the Ministry of Food has issued a new code of rules stating that a veterinary surgeon and two meat inspectors have to be included in the company of every whaling factory ship.

Their duties are to watch over the killing of the whales, inspect the meat at every stage of its preparation, and certify that it conforms to the hygienic conditions laid down.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.

Correspondents are requested to write as briefly as possible.

HARD PAD DISEASE

Sir,—Hard pad condition in dogs of all ages has been encountered in the month of December, 1948, in this country. I presume it has been imported along with dogs from the United Kingdom.

Up to the time of writing, I have only encountered the condition in and around the capital, Salisbury.

Yours faithfully,

David Burgess,
Major, late R.A.V.C.

19, North Avenue,
Salisbury, S. Rhodesia.
January 3rd, 1949.

DISPOSAL OF ANIMAL CARCASES

Sir,—The attention of the Executive Committee of this Association has been drawn to the correspondence upon the subject of knackers' yards which has recently appeared in your journal.

Whilst there is a certain measure of justification in the comments regarding the insanitary conditions of some knackers' yards, the responsibility for this condition must ultimately rest upon the local authority which grants the licence and thus permits the perpetuation of conditions which are properly described as disgraceful.

Under the Food and Drugs Act, 1938, the local authority is responsible for the licensing of knackers' yards and before granting such a licence is bound to send one of its officers to inspect the premises concerned. Where the application for the grant of a licence is by way of renewal of an existing licence, there is no legal obligation upon it to insist upon prior inspection, but the absence of a legal obligation surely does not in any way lessen the public duty imposed upon the local authority.

If either the premises are unsuitable or the applicant is not a proper person to hold such a licence, the local authority has power to, and in the opinion of the Association should, refuse such a licence.

Some doubt exists as to what the expression "fit and proper person" means but this Association's view is that a person who does not keep his premises in a sanitary condition cannot be said to be a proper person to hold a knacker licence.

Knackers' yards can be divided into two kinds; those which possess processing plants and produce high grade protein feeding meals and organic fertilisers and the smaller ones which do not.

The processing knackerman utilises his material partly to produce the meals and fertilisers which are in short supply and in doing so he performs the duty which it is suggested should be carried out by the municipal concern, and clean and fresh flesh is sold to the public as feeding-stuffs for cats and dogs for which there is a great popular demand.

The smaller knackerman sells the flesh from his yard (when it is in a fit condition to sell) to the public and disposes of the rest of the material to manufacturers who treat and work it up in various ways.

Members of this Association generally are on the best of terms with veterinary surgeons practising in the neighbourhood of their yards and gladly provide facilities for *post-mortem* examinations although, of course, a knackers' yard does not possess all the facilities and equipment customarily found in the dissecting room.

May I suggest that members of the veterinary profession desiring facilities for dissection should, at any rate in the first instance, approach knackermen trading in their vicinity and enquire if they will extend them the facilities desired.

Before the war there were some 190 knackers' yards in England and Wales. The number has now grown to over 400 and in the Association's opinion, much of the criticism directed towards the Knacker Industry is attributable to the activities of newcomers into a trade which they do not properly understand.

Yours faithfully,

ALFRED COOPER, *Chairman.*

The Licensed Horse Slaughterers Association,
174, Gresham House,
Old Broad Street, E.C.2.
February 3rd, 1949.

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MEAT HYGIENE

Sir,—We have to thank Mr. Anthony for his letter of January 28th. We were not unaware of the 1934 Report to which he doubtless refers but we would add that, in addition to our opinions, we were taking into consideration views expressed by the American Meat Institute as recently as 1948.

Yours faithfully,

A. ROBERTSON,
A. GINSBERG.

Department of Veterinary Hygiene,
Royal (Dick) Veterinary College,
Edinburgh, 9.
February 4th, 1949.

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UNIVERSITY QUALIFYING DEGREES

Sir,—I learn with dismay, from the announcement appearing in your current issue, that the University of Bristol proposes to offer a B.V.Sc. degree for graduates from its recently founded veterinary school.

This question of qualifying degrees seems to me of some importance, though I believe no comment on it has appeared in *The Veterinary Record* since you published a letter of mine about two years ago.

While not wishing to disparage the merits of the B.Sc. and B.V.Sc. degrees as ancillary qualifications, which serve a useful purpose under the existing system of veterinary education, it is my belief that, even to the informed public, they are not sufficiently distinct from B.Sc. degrees in pure science, which are awarded after three or, exceptionally, four years' work. It is not my intention to canvass for the grant of a doctorate as a primary qualification; indeed, British university tradition makes it most unlikely that this suggestion would receive consideration. However, a B.V.M. or V.M.B. degree would be at once distinctive and adequate; fitting qualification for a graduate on completion of the protracted course of study necessary to gain admission to the profession.

As a result of the 1948 Act, the public will gradually become familiar with the new qualifications required for registration as a veterinary surgeon. Such a favourable opportunity for instituting this reform will not recur.

The Council of the Royal College could act only in an advisory capacity on this matter, but I do urge that they should employ their full influence to impress the merits of this claim on the universities concerned. Even in the case that has led me to write this letter it is not too late to hope for success, since five years must pass before any students reach the point of graduation.

Yours faithfully,

THOMAS M. LEACH.

63, Hilders Road,
Western Park, Leicester.
January 31st, 1949.

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CINEMA CONTACT FACILITY

Sir,—Following an argument with the manager of the local A.B.C. cinema who at first refused to contact me in the audience for a calving case and afterwards made it quite clear that he would not carry out such a request again, I wrote to the chief of the theatre department and I thought it might interest members to read this extract from his reply:—

"However, I am to-day issuing instructions to all our managers to include veterinary surgeons in the compulsory category and you may rest assured, for the future, that should your services be required you will be contacted. In order to facilitate such contact, will you be kind enough to leave your name at the box office or with an attendant when you go in so that, if an enquiry is made, they will know you are in the theatre. A slide will then be put on the screen, asking you to go to the entrance hall."

It seems at last that the veterinary surgeon is beginning to stand for more than, "the man 'wot' puts dogs to sleep"—which was the last description applied to me.

Yours faithfully,

SIDNEY S. NYE.

230, South Street,
Romford.
February 2nd, 1949.

DISEASES OF ANIMALS ACTS, 1894 TO 1937, AND AGRICULTURE ACT, 1937 (PART IV).

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period	Anthrax	Foot-and-mouth	Fowl Pest	Parasitic Mange*	Sheep Scab	Swine Fever
Total 1st to 15th January 1949 ...	12	—	15	—	3	1
Corresponding period in—						
1948 ...	3	—	37	—	6	2
1947 ...	1	20	—	—	4	2
1946 ...	9	4	—	1	3	19

NOTE.—The figures for the current year are approximate only.
* Excluding outbreaks in Army Horses.

Tuberculosis Order of 1938

The number of bovine animals slaughtered under the Tuberculosis Order of 1938 during the period October 1st to December 31st, 1948, was 1,386.